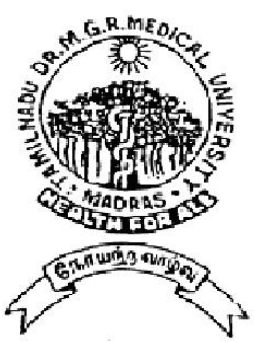
A COMPARISON OF SUBARACHNOID BLOCKADE IN PRE-ECLAMPSIA PATIENTS AND NORMAL PREGNANT PATIENTS COMING FOR CAESAREAN SECTION

A STUDY OF 84 CASES

DISSERTATION SUBMITTED FOR

DOCTOR OF MEDICINE

BRANCH X (ANESTHESIOLOGY)



THE TAMILNADU DR.M.G.R. MEDICAL

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MARCH 2010

CERTIFICATE

This is to certify that this dissertation entitled **"COMPARISON OF SUBARACHNOID BLOCKADE IN PREECLAMPTIC PATIENTS WITH NORMAL PREGNANT PATIENTS FOR CAESAREAN SECTION"** submitted by **DR. S.YOGALAKSHMI** to the faculty of ANAESTHESIOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement in the award of degree of M.D. Degree, Branch -X (ANAESTHESIOLOGY), for the March 2010 examination is a bonafide research work carried out by her under our direct supervision and guidance.

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INTRODUCTION

Eclampsia is the term coined by Hippocrates in 4th century BC . This greek term eclampsia means sudden development. Preeclampsia is a night mare to obstetrician and also obstetric anesthesiologist because of the danger it imposes both to mother and fetus. Selection of spinal anesthesia for severely preeclamptics patients requiring cesarean section is controversial. In 1985 William in his text book of obstetrics recommended avoiding regional anesthesia because of the fear of sudden, severe hypotension induced by splanchnic blockade, immediate danger from pressor agents and subsequent danger from large volume of fluid given to correct hypotension. In early 1990's several well conducted studies contradicted this doctrine . With the recent availability of PCWP monitoring and Thromboelastography, Regional anesthesia again came to play and it was accepted by many obstetric anesthesiologists. These studies made us to implement spinal anesthesia in preeclamptic patients and results of the study were discussed in detail in this dissertation.

AIM OF THE STUDY

Our aim is to understand the following physiological variants in preeclamptic patients in comparison to normal pregnant patients coming for cesarean section under Subarachnoid blockade like

1. Hemodynamic variables
2. Vasopressor requirement
3. Total intravenous fluid requirement
4. Vasopressor requirement
5. Neonatal out come
6. Postoperative hemodynamic stability

HISTORY OF OBSTETRIC ANAESTHESIA

The position of woman in any civilization is an index of the advancement of that civilization.The position of woman is gauged best by the care given to her at the birth of her child. JAMES YOUNG SIMPSON – JANUARY 1847 used Diethyl ether to anesthetise a woman with a deformed pelvis for delivery. Simpsons most articulate and persuasive critic was CHARLES . D . MEIGS PROFESSOR OF MIDWIFERY at Jeffersson medical college . He questioned the safety of anesthesia on fetus and believed that any drug that abolishes pain will alter uterine contraction. JOHN SNOW anesthetized the Queen for her eighth delivery. This changed the reaction of public towards etherisation for child birth.

Next major innovation in obstetric anesthesia came approximately 50 years later. DAMMERSCHALFF - which means twilight sleep was a technique developed by VONSTEINBUCHEL and popularized by GAUSS of Freiberg . It combined opioids with scopolamine to make women amnestic and somewhat comfortable during labour. In 1853 - hollow metal needle and syringe was developed .This technical advance simplified administration of opioids and facilitated the development of twilight sleep.

THE EFFECTS OF ANESTHESIA ON THE NEW BORN

The idea that gases crosses placenta appeared long before the discovery of oxygen and carbondioxide. JOHN SNOW observed depressed neonatal breathing and motor activity and smelled ether on the breath of neonates delivered from mothers who had been given ether.

IN 1953 – APGAR described a simple reliable system for evaluating newborns and showed that it was sufficiently sensitive to detect differences among neonates whose mothers had been anesthetized for cesarean section by

different techniques. Infants of woman with spinal anesthesia had higher scores compared to general anesthesia.

THE EFFECTS OF ANAESTHESIA ON LABOUR

MEIGS said that Etherisation suppressed uterine function.

JOHN SNOW withheld anaesthesia until the second stage of labour and limited administration of drugs to brief periods during contraction.

Regional anesthesia appeared during the first decades of twentieth century. The first papers describing obstetric applications of spinal ,lumbar, epidural, caudal, paravertebral, parasacral, pudendal nerve block appeared between 1900 and 1930. Recognition of the potential effects of Regiona anesthesia on labour developed more slowly and primarily because obstetrician seldom uses it. Chief advantage of regional anesthesia – the relative absence of drug effect on infant was not recognized by obstetricians. CLELAND described his experience with paravertebral anesthesia , but he also wrote athoughtful analysis of nerve pathways mediating labour pain. Continuous caudal anesthesia introduced by HINGSON AND EDWARDS in which a malleable needle remained in the sacral canal throughout labour. Small flexible plastic catheters eventually replaced malleable needles and made continuous epidural anesthesia even more popular.

Ironically natural childbirth appeared just as regional anesthesia started to become popular and clinicians began to understand how to use it without disrupting labour progression.

CLASSIFICATION OF PREECLAMPSIA

Working group of National high blood pressure education programme classified hypertension occurring during pregnancy as follows[18]

1. Gestational Hypertension. (Formerly pregnancy induced hypertension or transient hyperstension)
2. Preeclampsia.
3. Eclampsia.
4. Preeclampsia superimposed on chronic hypertension.
5. Chronic hypertension.

1) GESTATIONAL HYPERTENSION :-

It is defined as systolic BP of greater than 140 mmHg and diastolic BP greater than 90 mmHg for the first time during pregnancy without Proteinuria. The increase in BP return to normal within 12 weeks postpartum. So the final diagnosis is made only in the postpartum period. Patient may have other signs of preeclampsia for example epigastric discomfort or thrombocytopenia.

2) PREECLAMPSIA

The term preeclampsia describes the development of hypertension with proteinuria after 20 weeks gestation. It is classified into two types.

1) Mild preeclampsia

2) Severe preeclampsia

MILD PREECLAMPSIA :

It is defined as BP of greater than 140/90 mmHg after 20 weeks gestation with proteinuria greater than 300mmHg /24 hours.

SEVERE PREECLAMPSIA

It is defined as BP greater than 160 mmHg systolic or greater than 110

mmHg diastolic on two occasions atleast 6 hours apart while the patient is on bed rest with any of the following features of severity .

FEATURES OF SEVERE PREECLAMPSIA:

1. Serum creatinine > 1.2mg/dl
2. Platelets < 100000/mm3
3. Microangiopathic hemolysis
4. Elevated ALT/AST
5. Persistent headache
6. Persistent epigastric pain

INDICATIONS OF SEVERITY:-

Abnormality	Mild	Severe
Diastolic B.P	< 100 mmHg	110 mmHg or higher
Proteinuria	Trace to 1+	Persistent 2+ or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion	Absent	Present(eclampsia)
Laboratory results:		
Serum Creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Elevation of Liver	Minimal	Marked
enzymes	Absent	Obvious
Fetal growth restriction	Absent	Present
Pulmonary edema	Absent	Present

3) ECLAMPSIA:

Eclampsia is occurrence of seizures in a woman with preeclampsia that cannot be attributed to other causes. Seizures are grand mal type which may appear before, during or after labour.

4) PREECLAMPSIA SUPERIMPOSED UPON CHRONIC HYPERTENSION:-

It is defined as new onset proteinuria ≥ 300 mg / 24 hours in hypertensive women but no proteinuria before 20 weeks gestation or a sudden increase in proteinuria or blood pressure or platelet count < 100,000 / mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

5) CHRONIC HYPERTENSION :

The diagnosis of chronic underlying hypertension is suggested by.

1) Hypertension (140/90mmhg or greater) antecedent to pregnancy.
2) Hypertension (140/90mmhg or greater) detected before 20 weeks (unless there is gestational trophoblastic disease).
3) Persistent hypertension long after delivery.

Additional factors supporting diagnosis:-

a) Multiparity and hypertension complicating pervious pregnancy other than the first.
b) Strong family history of essential hypertension.

UNDER LYING CHRONIC HYPERTENSIVE DISORDERS:

1) Essential familial hypertension.
2) Arterial abnormalities.
    a) Renovascular hypertension
    b) Coarctation of aorta.
3) Endocrine disorders.
    a) Diabetes
    b) Cushing's Syndrome
    c) Primary Aldosteronism
    d) Pheochromocytoma
    e) Thyrotoxicosis.
4) Glomerulo nephritis
5) Reno prival hypertension.
    a) Chronic glomerulonephritis
    b) Chronic renal insufficiency
    c) Diabetic nephropathy
6) Connective tissue diseases :
    a) Systemic lupus erythematosus
    b) Scleroderma
    c) Polyarteritis nodosa.
7) Polycystic kidney disease
8) Acute renal failure
9) Obesity.

EPIDEMIOLOGY

It is the third highest cause of direct maternal death. Incidence varies among different countries or populations . It is 5% according to (Williams Obstetrics) 22nd edition and 8 – 10% (Mudaliar menon) Tenth edition.

RISK FACTORS

Risk factors for preeclampsia is given under the following headings.

I. Partner related

1. Nulliparous / primi / teenage
2. Limited sperm exposure / donar inseminisation / oocyte donation
3. Oral sex (risk reduction)
4. Partner who fathered

   Preeclamptic pregnancy in another women.

II. Non partner related

1. H/O previous preeclampsia
2. Age,interval between pregnancy

III. Presence of specific underlying disorder

1. Chronic HT , Renal disease
2. Obesity, Insulin resistance, low birth weight, gestational diabetes milletus, Type I Diabetes milletus,
3. Activated protein - C resistance, protein – S deficiency.
4. Anti phospholipid antibodies
5. Hyper homocystenimia : sickle cell disease

IV. Exogenous

1. Smoking
2. Environmental factors – high altitude
3. In utero diethyl stilbestrol exposure.
4. Poor socioeconomic status .

PATHOPHYSIOLOGY

Preeclampsia is a multisystem disorder that primarily affects the maternal cardiovascular , central nervous system and genitor urinary system. However all systems may be involved to some degree.[21]

I. CARDIOVASCULAR CHANGES:-

a) In Gestational Hypertension there is increase in Cardiac output and decrease in peripheral vascular resistance.

b) In preeclampsia the Cardio Vascular changes were classified by Cotton et al as follows.

1. Increased Cardiac output, Normal or increased systemic vascular resistance, Normal or slightly decreased blood volume and filling pressures.
2. Normal Cardiac output, increased systemic vascular resistance, decreased filling pressure.
3. Increased systemic vascular resistance, decreased blood volume, decreased Left ventricular function.

Three factors may explain these differences

1) Women with preeclampsia might present with a spectrum of cardiovascular findings dependant upon both severity & duration.
2) Chronic underlying disease may modify the clinical presentation.
3) Therapeutic interventions may alter these findings.

Three Separate groups based on Clinical Management

1) No therapy for preeclampsia.

2) Magnesium sulfate and Hydralazine without large volumes of intravenous fluid.
3) Magnesium sulfate and Hydralazine plus intravenous volume loading.

COLLOIDAL ONCOTIC PRESSURE:

Colloidal oncotic pressure is already decreased in normal pregnancy because of hypoalbuminemia , often is further decreased in preeclamptic women. This promotes increased risk for pulmonary edema .

COLLOIDAL ONCOTIC PRESSURE IN VARIOUS STAGES OF PREGNANCY

	Antepartum	Postpartum
Normal pregnancy	22mmHg	17 mmHg
Preeclampsia	18mmHg	12mmHg

SEVERE PREECLAMPSIA AND ECLAMPSIA:-

ASSOCIATED HEMODYNAMIC MEASUREMENTS:-

This is a study showing comparison of various hemodynamic variables before starting therapy and after initiating therapy by various authours. [23]

Therapy	No	C.O (L/min)	PCWP (mmhg)	Left Ventricular Stroke work index ($g.m.m^2$)	Systemic vas. Resistance (dyne / sec) Per cm^{-5}
1. Before Therapy					
a.Groenendijk etal.(1984)	10	4.66	3.3	44	1943
b.Visser&wallenbeg (1995)	87	$(3.3)^6$	7	NA	3003
With therapy 2. Magnesium, Hydralazine fluid restriction					
a. Benedicts etal (1980)	10	7.4	6.0	82	1322
b. Hankins etal (1984)	8	6.7	3.9	66	1357
3.Magnesium, Hydralazine volume expansion.					
a. Rafferty with Berkowitz (1980)	3	11.0	7.0	89	780
b. Phelan and yurth (1982)	10	9.3	16.0	89	1042

BLOOD VOLUME CHANGES

Pouta et al demonstrated that an intravenous fluid bolus resulted in a greater increase in CVP in preeclamptic women than in normotensives.[19]

Tabular column showing the blood volume changes

Variables	Eclampsia	Non pregnant	Normal pregnant
1)Blood Volume (m1)	3530	3035	4425
2)Change (%)	+16		(+47)
3)Hematocrit	40.5	38.2	34.7

* Hemo concentration is a hallmark of eclampsia.
* Woman with eclampsia therefore is unduly sensitive to vigorous fluid therapy administered in an attempt to expand the contracted blood volume to normal pregnancy levels. She is sensitive as well to even normal blood loss at delivery.

HEMATOLOGICAL CHANGES:-

Well documented coagulation abnormalities occur in patients with preeclampsia.

They are as follows

* Thrombocytopenia.
* Reduction in clotting factors
* Erythrocytes are traumatised and bizarreshaped. They undergo rapid hemolysis because of altered red cell membrane

Coagulation disturbances leading to hypercoagulability is listed below

* Fibrin degradation products increased
* Plasma fibrinogen – decreased
* Fibirnogen decreased
* Fibrinopeptide A increased
* Anti thrombin III decreased
* Factor IX, X, XI, XII decreased
* Fibrin monomer – positive
* Thrombin time – prolonged.

THROMBOCYTOPENIA:-

Burrows et al found elevated platelet associated immunoglobulin G in 35% of preeclamptic patients which suggest that an immune mechanism is probably responsible for the thrombocytopenia Platelets demonstrate a 50% reduction in sensitivity to PGI2

CAUSES:-

* Despite of increased platelet production, platelet activation and platelet consumption process is increased.

* Thrombopoietin level is increased - a cytokine that promotes megakaryocytes proliferation.

Evidence of platelet activation include

1) Increased release of beta – thromboglobulin by platelets

2) Appearanceof megathrombocytes (giant platelets) in peripheral blood

3) Count < 100 000 ml – severe disease.

After delivery platelet count increased progressively to reach a normal level in 3 days.

ERYTHROCYTE:-

Red blood cell undergoes the following changes

* Hemolysis
* Schistocytosis, spherocytosis, reticulocytosis
* Hemoglobinuria & occasionally hemoglobinemia - Changes due to increased erythrocyte membrane fluidity.

OTHER CLOTTING FACTORS:-

* Antithrombin III –decreased
* Fibronectin – a glycoprotein associated with vascular endothelial cell basements membrane is elevated in women with preeclampsia.

ENDOCRINE AND METABOLIC CHANGES:-

Renin angiotensin aldosterone system (RAAS) undergoes complex pathological changes which is tabulated below.[32]

ENDOCRINE CHANGES:-

This tabular column depicts the changes in Renin Angiotensin Aldosterone system.

	Normal Pregnancy	**Preeclampsia**
Renin	Increased	Decreased
Angiotensin II	Increased	Decreased
Aldosterone	Increased	Decreased

PREECLAMPSIA:-

Sodium retention / Hypertension

↓

Renin secretion by Juxta glomemrular cells decreases

↓

Angiotensinogen → Angiotensin I → Angiotensin II
(decreases) (decreases)

↓

Aldosterone (decreases)

* Deoxy corticosterone (DOC) increased in third trimester
* Atrial natriuretic peptide (ANP) increased in women with preeclampsia . It is vaso active and promotes sodium and water excretion by inhibiting Aldosterone , Renin activity, Angiotensin II and Vasopressin.
* Increase in ANP causes increased cardiac output and decreased Peripheral vascular resistance in response to volume expansion.

FLUID AND ELECTROLYTE CHANGES:

Increase in volume of ECF

MECHANISM:-

Endothelial injury causing proteinuria and Decreased plasma oncotic pressure causing filtration imbalance .This leads to displacement of intra vascular fluid to interstitium.

KIDNEY:

The characteristic renal pathology is glomerular enlargement with resultant ischemia.

Parameters	Renal blood flow	Glomerular filtration rate	Plasma uric acid
Normalpregnancy	Increased	Increased	Normal
Preeclampsia	Decreased	Decreased	Increased

Various results of the studies regarding renal vascular system in preeclampsia is given below.

* Williams & Chesley 1945- decreased creatinine clearance
* (Taufield & associates 1987) decrease urinary excetion of Calcium because of increase tubular reabsorption.
* Pritchard & colleagues (1984) – increase Plasma creatinine (upto 2-3mg / dl)
* Lee & associates (1987) □ oliguria despite normal left ventricular filling pressures and Urine sodium concentration increased.
* Krishnan & co workers (1988) infused i.v dopamine into oliguric women with preeclampsia and found increased urine output,increased Fractional Na^+ excretion, increased free water clearance.

PROTEINURIA:-

1. Meyer & colleagues (1994) – proteinuria quantification by dipstick was not accurate and recommended 24 hour measurements.

2.Adelberg & coworkers (2001) – urine protein quantification in 8 and 12 hour

samples correlated when there was mild to moderate proteinuria.

3. Neihardt colleagnes (2002) & Rodriguez Thompson & Lieberman (2004)

Found urinary protein creatinine ratio to predict significant proteinuria is good / suitable.

WHAT IS PROTEINURIA?

Increased permeability to most large molecular weight proteins thus abnormal albumin excretion accompanied by other proteins such as hemoglobin, globulins and transferrin.

ANATOMICAL CHANGES:

Using Light Microscopy Sheehan (1950) noted the following changes

1) Glomeruli were enlarged by 20%
2) Capillary loop were contracted
3) Endothelial cells are swollen

ELECTRON MICROCOPY:-

The following changes were noted

1) Glomerular capillary endotheliosis
   (by spargo & associates 1959).
2) The swollen endothelial cells block the capillary lumen.
3) Homogenous deposits of an electron dense substance are found between basal lamina and endothelial cells and also within endothelial cells.

IMMUNOFLOURESCENT STAINING:- showed deposits of fibrinogen in biopsy specimen. Deposits disappear in first week postpartum.

LIVER:-

Hepatic changes in women with fatal eclampsia (by virchow)

1) Regions of periportal hemorrhage in liver periphery.
2) Sheehan & lynch (1973) found infarction accompanied haemorrhage in 50% cases.
3) Elevated serum hepatic transaminase levels (Pritchard & colleagues) 1954.
4) Subcapsular hematomas - patient presents with epigastric or subcostal pain caused by stretching of liver capsule
5) Hepatic rupture

BRAIN:-

Anatomical pathology: - Two types

1) Gross Haemorrhage due to rupture of arteries caused by severe hypertension.
2) Haemorrhage – may be wide spread or focal haemorrhagic lesions with edema, hyperemia, ischemia, thrombosis.

CEREBRAL BLOOD FLOW:-

Non invasive transcranial doppler ultrasonography - Measures Cerebral blood flow. Thereby cerebral perfusion pressure is calculated.

Belfort associates (1999)

Preeclampsia is associated with increased cerebral perfusion pressure counter balanced by increased cerebral vascular resistance and no net change in cerebral blood flow.

Eclampsia Loss of auto regulation of CBF leading to hyper perfusion.

BLINDNESS:-

Otherwise Called Amaurosis (Greek work – dimming) Follows eclamptic convulsion in 10% women. CT scan shows extensive occipital lobe vasogenic edema. Blindness develop upto a week or more following delivery. Blindness lasts from 4 hours to 8 days but it resolved completely

inall cases.

RETINAL DETACHMENT:-

May also cause altered vision, usually one sided. Prognosis is usually good Vision usually returns to normal within a week.

PERMANENT VISUAL DEFECTS:-

Permanent visual loss is due to Cerebral infarction or retinal artery ischemia and infarction.

CEREBRAL EDEMA:-

Patients with cerebral edema presents with Lethargy , Confusion , Blurred vision, Coma. Symptoms are waxing & waning in nature.

EEG :- Abnormalities develop within 48 hrs of seizures. Half of abnormalities persisted longer than 1 week but most resolved in 3 months.

UTERO PLACENTAL PERFUSION:-

In preeclampsia there is Vasospasm of uterine vessels leading to decreased uteroplacental perfusion which leads on to increased Perinatal morbidity and mortality. [11]

	Normal Pregnancy	Preeclampsia
Mean Diameter of Maternal myometrial Spiral arterioles	500 micrometer	200micrommeter

INDIRECT MEASUREMENTS:-

Conversion of 17B estradiol to dehydro androsteronesulfate occurs in placenta and clearance of 17B estradiol is accurate reflection of human placental perfusion.Normal placental clearance rate increases with advancing pregnancy. In preeclampsia placental clearance decreases.

DOPPLER VELOCIMETRY:-

Measurement of blood flow velocity through uterine arteries used to estimate resistance to uteroplacental blood flow. Vascular resistance is estimated by comparing arterial systolic and diastolic velocity waveforms. During normal pregnancy , uteroplacental bed constitutes a low resistance circuit, and Doppler waveforms demonstrate continuous diastolic flow. In preeclampsia downstream resistance increases, diastolic velocity decreases, systolic/diastolic velocity ratio increases.

ETIOLOGY

Hypertensive disorders due to pregnancy are more likely to develop in women who

1) Are exposed to chorionic villi for the first time.
2) Are exposed to a super abundance of chorionic villi as with twins or hydatidiform mole.
3) Have preexisting vascular disease.
4) Are genetically predisposed to hypertension developing during pregnancy.

Sibai studied plausible potential causes as [31]

1) Abnormal trophoblastic invasion of uterine vessels.
2) Immunological intolerance between maternal & fetoplacental tissues.
3) Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.
4) Dietary deficiencies.
5) Genetic influences.

ABNORMAL TROPHOBLASTIC INVASION:-

- In normal implantation the uterine spiral arteries undergo extensive remodeling as they are invaded by endovascular trophoblasts.
- In preeclampsia there is incomplete trophoblastic invasion.
- Decidual vessels but not myometrial vessels are lined with endovascular trophoblasts.

ELECTRON MICROSCOPY: - [30]

Electron microscopy reveals the following changes

1. Endothelial damage and insudation of plasma consituents into vessel walls, proliferation of myointimal cells and medial necrosis.
2. Lipid accumulates in myointimal cells and then in macrophages leading to atherosis.
3. Obstruction of spiral arteriolar lumen by atherosis causing Impaired placental blood flow which in turn causes preeclampsia syndrome

IMMUNOLOGICAL FACTORS:-

1. Labarrer 1988 - Maternal placental interface shows microscopic changes suggestive of acute graft rejection.
2. Risk of preeclampsia - decreased formation of blocking antibodies to placental antigens. Various possible etiological theories are listed below
3. In First pregnancy there is increased risk because effective immunisation by previous pregnancy is lacking.
4. In Multiple pregnancy the number of antigenic sites provided by placenta unusually great. (Beer 1978)
5. In Abortion prior immunisation does not occur. (Crick land & associates)
6. Immune maladaptation - Dekker sibai (1998)
7. Decreased Helper T cells - Bardegueze associates (1991)
8. Th1/Th2 imbalance (t-helper cells1/t helper cells2) - Yoneyama coworkers
9. Th2 predominance may be mediated by adenosine in preeclampsia.

THE VASCULOPATHY AND THE INFLAMMATORY CHANGES:-

Redmann and colleagues (1999) found that endothelial dysfunction associated with preeclampsia can result from “a generalized perturbation of the normal. Generalized maternal intravascular inflammatory adaptation to pregnancy.”

Cytokines like TNF alpha and interleukins are released during oxidative stress which in turn produces reactive oxygen species and free radicals. These toxic radicals modify Nitric oxide production, interfere prostaglandin imbalance and injure endothelial cells. [22]

Treatment with Antioxidants is useful.

EXAMPLES OF ANTIOXIDANTS:-

They include Vitamin E (Alpha - tocopherol), Vitamin C (Ascorbic acid), Beta Carotene.

NUTRITIONAL FACTORS:-

Studies have shown that supplements of zinc, calcium, magnesium can prevent preeclampsia,

Vitamin C deficiency increases the incidence of preeclampsia.

In Obesity there is increased production of C - reactive protein an inflammatory marker which is associated with increased incidence of preeclampsia

GENETIC FACTORS:-

1) Kipatrick associates (1989) found an association between HLA – DR4 and proteinuric hypertension.
2) Half and associates (1992) revealed that maternal humoral response directed against fetal anti–HLA–DR immunoglobulin antibody might

influence the development of gestational hypertension.

3) FactorV leiden mutation consists of an aminoacid substitution of glutamine for arginine at position 506 in the factor v molecule causing thrombosis of maternal myometrial vessels.

4) ANGIOTENSINOGEN

A molecular variant of angiotensinogen(angiotensinogen gene T 235) has been described in association with preeclampsia. It increases the risk of developing preeclampsia by a factor of 20.[33]

Women heterozygous for Angiotensinogen gene variant T 235 is associated with increased incidence of preeclampsia & fetal growth restriction.

PATHOGENESIS:-

VASOSPASM:-

The concept of vasospasm was advanced by volhard (1918) based on direct observation of small blood vessels in nail beds, ocular fundi and bulbar conjunctiva. Endothelial cell damage causes interstitial leakage causing Platelets and fibrinogen to be deposited sub endothelialy. [22]

ENDOTHELIAL CELL ACTIVATION:-

* Endothelial cell activation has become the center piece in the contemporary understanding of the pathogenesis of preeclampsia.

* Intact endothelium has anticoagulant properties and it also blunts the response of vascular smooth muscle to agonists by releasing Nitric Oxide.

* Damaged endothelial cell secretes substances that promote coagulation & increased the sensitivity to vasopressors.

INCREASED PRESSOR RESPONSES:-

Normally, pregnant women develop refractoriness to infused vasopressors.

Women with preeclampsia have increased vascular reactivity to infused norepinephrine & angiotensin II . [4,33]

PROSTAGLANDINS:-

1. PG I_2 production decreased in preeclampsia which promotes platelet aggregation
2. Thromboxane A_2 increase also promotes platelet aggregation
3. Prostacyclin, Thromboxane A_2 ratio decreases and net result is increased sensitivity to angiotensin.

NITRIC OXIDE:

1. Nitric oxide is a potent vasodilator synthesized from L-Arginine by endothelial cells.
2. Inhibition of Nitric Oxide synthesis increases MAP, decreases Heart rate and reversed the pregnancy induced refractoriness to vasopressors.
3. Nitric oxide is the compound that maintains the normal low pressure vasodilated state characteristic of fetoplacental perfusion.
4. Nitric oxide is also produced by fetal endothelium
5. Increased in 1. Preecalmpsia 2. Diabetes 3. Infection .
6. Nitric oxide production is increased in response to a compensatory mechanism for the increased synthesis and release of vasoconstrictor and platelet aggregating agents.

ENDOTHELINS:-

Endothelin is a potent vasoconstrictor. It is Produced in endothelium. Plasma Endothelin I is increased in Normotensive pregnant women. In preeclampsia there is marked increase in plasma endothelin 1. Source of plasma endothelin 1 is systemic endothelial activation. Treatment with Magnesium sulfate decreases Endothelin 1 concentration.

ANGIOGENIC FACTORS:

1.VEGF – Vascular Endothelial Growth Factor. Increased in normal pregnancy

2. PIGF – Placental Growth Factor

FUNCTIONS:

a) Promote angiogenesis

b) Induce Nitric oxide and vasodilatory prosta glandin

VEGF is markedly increased in women with preeclampsia but its bioavailability is decreased.

PREDICTION AND PREVENTION

PREDICTION:-

1. ROLL OVER TEST: Gant & colleagues (1974)

A hypertensive response induced by having a women at 28 – 32 weeks assume the supine position after lying laterally recumbent predicted gestational hypertension. Women with positive roll over test is also found abnormally sensitive to infused angiotensin II.

2. URIC ACID:-

Elevated serum uric acid levels due to decreased renal urate excretion found in woman with preeclampsia. Jacobson & Colleagues (1990) – found that Plasma uric acid values > 5.9 mg /dl at 24 weeks has positive predictive value of 33% for preeclampsia. Weerasekera & petris (2003) found no significant increase in serum uric acid levels.

3. FIBRONECTIN:-

Endothelial cell activation is likely the cause for elevated serum cellular fibronectin levels.

4. COAGULATION ACTIVATION:-

* Thrombocytopenia and platelet dysfunction are integral features of preeclampsia.
* Increased destruction causes platelet volume to increase.
* High platelet volume denotes impending preeclampsia.
* In normal pregnancy fibrinolytic activity decreased due to increase in plasminogen activator inhibitor 1 and 2.
* In Preeclampsia plasminogen activator inhibitor (PAI 1) increase relative to PAI 2 because of endothelial dysfunction.

5. OXIDATIVE STRESS:-

Increase in lipid peroxides and decrease in antioxidants causes preeclampsia.

Lipid peroxides : Malondialdehyde.

Pro oxidants : Iron, transferrin, ferritin, blood lipids including triglycerides, freefatty acids & lipoproteins.

Anti Oxidants : Vitamin – C & Vitamin – E

Increased serum homocysteine in mid pregnancy shows 4 fold increase risk of preeclampsia. Homocysteine levels are influenced by folic acid supplementation.(Power & associates 2003)

CYTOKINES:-

Interleukins and TNF Alpha increased.

PLACENTAL PEPTIDES: Certain hormones are increased in preeclampsia which is given below.

MARKERS OF PREECLAMPSIA: 1 Corticotropin releasing hormone, Chorionic gonadotropin, Activin – A**,** Inhibin – A

FETAL DNA:-

Identification of fetal DNA in maternal serum may be predictive of preeclampsia.

UTERINE ARTERY DOPPLER VELOCIMETRY:

Measurement of ultra placental vascular resistance during doppler ultra sound evaluation of uterine artery impedance in second trimester.

PREVENTION:-

DIETARY MANIPULATION:

Knuist & Colleagues (1998) showed that sodium restricted diet is inefficient in preventing preeclampsia . Low dietary ca^{+} is associated with increased incidence of Gestational Hypertension. 14 randomised Trials with prenatal ca^{+} supplementation showed significant reduction in blood pressure in preeclamptics.

Fish oil capsules – to modify prostaglandin balance may be tried.

LOW DOSE ASPIRIN:-

60 mg aspirin to reduce the incidence of preeclampsia ineffective. Asprin suppress thromboxane suppression and thereby decreased platelet aggregation, increase prostaglandin-I2 production, also increased the incidence of placental abruption.

ANTIOXIDANTS: Chappel & colleagues found that Vitamin C (1000 mg / day) & Vitamin E (400 mg/day) versus placebo showed significant decrease in endothelial cell activation in preeclampsia.

EARLY PRENATAL DETECTION:-

1. Increase prenatal visits during 3rd trimester helps in early detection.
2. Women with BP > 140 / 90mmHg should be admitted and evaluated.
3. Women with diastolic BP > 81 – 89 mmHg with weight gain > 2 pounds / week in 3rd trimester were asked to visit at every 3-4 days interval.

INVESTIGATIONS

1. Hemoglobin and hematocrit
2. Platelet count
3. Quantification of protein excretion
4. Serum creatinine
5. Serum uricacid
6. Serum transaminase levels
7. Serum albumin
8. Serum lactic acid dehydrogenase levels
9. Blood smear
10. Coagulation profile

MONITORING

Monitoring of preeclamptic patients can be classified into two groups as follows

A) NON INVASIVE MONITORING:

Is required for all cases

1. Oxygen saturation
2. Automated blood pressure and pulse monitoring
3. Foleys catheter for urine output
4. Fetal heart rate monitoring.

B) INVASIVE MONITORING

1. Arterial line is mandatory for
    a) Morbidly obese woman
    b) Refractory hypertension where sodium nitroprusside is necessary
    c) Pulmonary edema where serial blood gas measurements are necessary
2. Central venous pressure monitoring is required for severa preeclamptic patients with oliguria
3. Pulmonary arterial line is mandatory if
    a) If initial CVP reading is high (8 or above)
    b) Oliguria persists even with normal central venous pressure
    c) Pulmonary edema
    d) Cardiovascular collapse

TREATMENT STRATEGY OF PREECLAMPSIA

ANTEPARTUM HOSPITAL MANAGEMENT:

1) Detailed examination followed by daily scrutiny for clinical findings such as headache, Visual disturbances, epigastric pain, rapid weight gain.
2) Weight on admittance and everyday thereafter.
3) Analysis for proteinuria on admittance every 2days there after.
4) BP readings in sitting position with an appropriate size cuff every 4 hours except between midnight and morning.
5) Measurements of Plasma or serum creatinine, hematocrit, platelets, serum liver enzymes.
6) Frequent evaluation of fetal size and amniotic fluid volume either clinically or by ultra sonography.
7) Reduced physical activity throughout the day
8) Ample but not excessive protein included is diet.

Further management depends upon.[14]

- Severity of preeclampsia, determined by presence or absence of conditions cited.
- Duration of gestation.
- Condition of cervix.

MANAGEMENT:-

BASIC MANAGEMENT OBJECTIVES:-

a. Termination of pregnancy with the least possible trauma to mother & fetus.
b. Birth of an infant who subsequently thrives.
c. Complete restoration of health to the mother.

TERMINATION OF PREGNANCY:-

Regardless of gestational age any of the following complications mandates immediate delivery

1. Severe hypertension that persists after 24- 48 hours
2. Progressive thrombocytopenia
3. Liver dysfunction
4. Progressive renal dysfunction
5. Premonitory signs of eclampsia
6. Evidence of fetal jeopardy.

DELIVERY IS THE CURE FOR PREECLAMPSIA.

Non stress test or Bio physical profile for fetal assessment measurement of lecithin sphingomyelin ratio in amniotic fluid is an evidence of lung maturity. Labour is induced by intravenous oxytocin, Cervical ripening with prostaglandin or osmotic dilator. If labour induction failed cesarean delivery.

ANTI HYPERTENSIVE DRUG THERAPY:-

Study		Prolongation pregnancy	Severe HT	Cesarean Delivery
200 pts. Sibai eta1	Labetatol (100)	20.1	5	36
	Placebo (100)	21.3	14	32
Study	Abruptio	Mean birth wt	Growth restriction	Neonatal breaths
Sibai eta1	2	2205	19	1
200 pts.	0	2260	9	0

“Growth restricted infants were twice as frequent in women given labetalol compared with those treated by hospitalization alone.

.

ECLAMPSIA

Preeclampsia complicated by generalized clonic tonic convulsions is termed Eclampsia .

COMPLICATIONS:-

1) Abruption of placenta (10%)
2) Neurological deficits (7%)
3) Aspiration Pneumonia (7%)
4) Pulmonary Edema (8%)
5) Cardio pulmonary arrest (4%)
6) ARF (4%)
7) Maternal death (1%)

CLASSIFICATIONS:-

1) Ante partum
2) Intra partum
3) Post partum

DESCRIPTION OF CONVULSIVE ATTACK:

1) Convulsive movements begin about the mouth in the form of facial twitchings.
2) After a few seconds the entire body become rigid in a generalized muscular contraction – phase 15 – 20 secs.
3) Suddenly jaws open & close violently & soon after the eyelids as well.

   Other facial muscles & then all muscles alternately contract & relax in rapid succession.

   So forceful are the contractions that woman may throw herself out her bed & if not protected. Her tongue is bitten by violent action of the jaws. This phase last about a minute.
4) Gradually the muscular movements become smaller and less frequent , finally woman lies motionless.
5) Throughout the seizure the diaphragm has been fixed, with respiration halted.

   For a few seconds the woman appears to be dying from respiratory arrest, but then she takes a long, deep stertorous inhalation & breathing is resumed.
6) Continuous convulsions – status epilepticus.
7) After a seizure coma ensues – overtime memories return.

* Respiration increased in rate 50 or more / min after a convulsion to combat

hypercarbia from lactic academia.

* cyanosis

* High fever is a grave sign for CNS hemorrhage.

Antepartum eclampsia labor may begin spontaneously after the convulsion ensue & progress rapidly.

If convulsions occur during labor contractions may increase in frequency and intensity & duration of labor may be shortened.

* Fetal bradycardia from maternal hypoxemia recovers in 3 – 5 minutes
* Pulmonary edema is caused by aspiration pneumonitis, from inhalation of gastric contents if vomiting accompanies convulsions.
* Cerebral hemorrhage is due to ruptured berry aneurysm or arteriovenous malformation.
* Blindness occurs in 10% of patient due to occipital lobe ischemia and retinal detachment.
* Psychosis lasts several days to 2 weeks.

“Until proved other wise all pregnant women with convulsion should be considered to have eclampsia.”

PROGNOSIS:-

Prognosis – 10 – 15% mortality rate

TREATMENT STRATEGY OF PREECLAMPSIA

MAGNESIUM SULPHATE THERAPY

DOSAGE SCHEDULE FOR SEVERE PREECLAMPSIA AND ECLAMPSIA:-

1) CONTINUOUS INTRAVENOUS INFUSION:-

a) Give 4 – 6 g loading dose of magnesium sulfate diluted in 100ml of IV fluid administered over 15 20 min.

b) Begin 2g/hr in 100ml of IV maintenance infusion.

c) Measure serum magnesium level at 4 – 6 hr and adjust infusion to maintain levels between 4 – 7 Meq / l.

d) Magnesium sulfate is discontinued 24 hr after delivery.

2) INTERMITTENT INTRA MUSCULAR INJECTIONS:-

a) Give 4gm of $Mgso_4.7H_2O$ USP as 20% solution intravenously at a rate not to exceed 1 gm / min.

b) Follow promptly with l0g of 50% $Mgso_4$ solution, one half (5g) injected deeply in upper outer quadrant of both buttocks through a 3 inch long, 20 guage neddle.

If convulsions persists after 6 min, give up to 2g more intravenously 20% solution at a ratr not to exceed l g/min.

3) Every 4 hr thereafter give 5g of 50% solution of $Mgso_4.7H_2O$ injected deeply in the upper outer quadrant of alternate buttocks.

Ensure that

a) Patellar reflex is present.

b) Respirations not depressed.

c) Urine output in the pervious 4 hour exceeded 100ml.

4) Magnesium sulfate is discontinued 24 hr after delivery.

Magnesium sulfate is not given to treat hyper tension.

PHARMACOLOGY & TOXICOLOGY OF MAGNESIUM SULFATE:-

* Magnesium sulfate USP is $Mgso_4.7H_2O$ & not $Mgso_4$.
* Parenteral Magnesium is cleared almost by renal excretion.
* Plasma Magnesium level to prevent eclampsia 4 – 7 Meq / l.
* Plasma Magnesium 10 Meq / l - patellar reflexes disappear presumably because of curarie form action.
* Plasma Magnesium > 10 Meq / l - respiratory depression.
* Plasma Magnesium > 12 Meq / l - Respiratory paralysis & arrest
* Antidote to $Mgso_4.H_2O$ is Calcium gluconate

Dose : 1gm IV

Action Short lived.

Patient with respiratory depression requires tracheal intubation and mechanical ventilation.

Benefcial effects of magnesium sulfate

1) It has an inhibitory effect at the neuro muscular junction.
2) Both invivo and invitro studies found magnesium to increase production of endothelial vasodilator prostacyclin.
3) Protects against ischemic damage of cells by substitution of calcium and also prevents entry of ca^{++} ions into ischemic cells.
4) Anticonvulsant by acting as a NMDA receptor (N – methyl – D – aspartate) antagonist

Effect of Increasing plasma magnesium levels:-

Plasma mg (meq/l)	Effects
1.5 – 2.0	Normal plasma level
4.0 – 8.0	Therapeutic range
5.0 – 10	ECG changes (PQ interval prolonged, QRS complex widens)
10	Loss of deep tendon reflexes.
15	Sinoatrial and atrioventricular block, respiratory paralysis.
25	Cardiac arrest

UTERINE EFFECTS:-

Mg^{++} ions in high concentrations may depress myocardial contractility both invivo and invitro.

FETAL EFFECTS:-

Magnesium crosses placenta and achieve equilibrium in fetal serum. Neonatal depression occurs only if there was severe hypermagnesemia at the time of delivery. Small but significant decrease in fetal heart rate variability.

MATERNAL MORTALITY IN ECLAMPSIA- TRIAL COLLABORATIVE GROUP MORTALITY.

Regimen	No	Maternal (%)	Perinatal (per 1000)
Magnesium sulfate	453	3.8	25
Diazepam	452	5.1	22
phenytoin	387	5.2	31

ANTI HYPERTENSIVE DRUG THERAPY:-

Rational:-

1) Prevent maternal morbidity associated with

a) Encephalopathy b) CVA c) End organ damage

Threshold for treatment of Diastolic BP is 105 – 110 mmHg

MAP – 125 mmHg

HYDRALAZINE TO CONTROL SEVERE HYPERTENSION:-

Hydralazine is given when Systolic BP > 160 mmHg and Diastolic BP >110 mmHg . Dosage: 5 – 10 mg doses is given at 15 – 20 min intervals until Diastolic BP decreased to 90 – 100mmHg. Fetal bradycardia is the expected complication . Hydralazine is the most commonly used drug. It Decreases episodes of acute blood pressure increases in pregnancy. It relaxes arterioles and decreases systemic vascular resistance through increased Cyclic AMP

Action:-

1) HR, SV, CO increased.

2) Increased uterine perfusion

LABETALOL :

It is an Alpha 1 and nonselective Beta blocker. (1:3 orally, 1:7 iv) .It is a second line drug. Decreases maternal SVR with out increased HR. Intravenous labetalol lowered BP more rapidy and associated tachycardia was minimal. Initial dose is 10mg then increased by 10 mg every l0 minutes NHBPEP (2000) recommends starting with 20 mg iv bolus then 40mg with in 10 minutes then 80 mg every 10 mins. Not to exceed 220 mg total dose per episode treated. Recent work – labetalol 1 mg / kg will decrease maternal blood pressure with out affecting the intervillous and fetal blood flow. Avoided in CHF / Asthma.

NITRO GLYCERINE:

* Rapid onset

* Produces abrupt hypotension

* Sublingual 400 microgram

Mechanism of action:

It produces Vascular smooth muscle relaxation by intracellular degradation to Nitric oxide , Increased C-Gmp and venous relaxation , decreased pre load .

NIFEDIPINE :-

Decreased influx of ca^{+} smooth muscles

Advantages:-

1) They act as vasodilator.
2) Uterine muscle relaxants.
3) Increased Renal Blood Flow.
4) It readily crosses placenta.
5) In severe preeclampsia it decreases maternal BP and prolongation of pregnancy and improvement of fetal oxygenation.

Nifedipine 10 mg oral dose repeated in 30 mints

Complications:-

I. Severe Refractory Hypertension: It can be treated with a) $Mgso_4$

b) IV labetalol & IV hydralazine.

SODIUM NITROPRUSSIDE

Nitroprusside is used unless there in no response to hydralazine, labetalol, nifedipine. Continuous infusion with dose of 0.25 micro gram / kg / min increased to 5 micro gram/ kg / min.

Mechanism of action:-

Sodium nitroprusside interact with sulph hydryl group on endothelium and releases Nitric oxide to relax arterial vessels.

Dose: 0.5 microgram / kg / min. Production of cyanide > 4 microgram / min

Fetal cyanide toxicity may occur after 4 hour.

Persistent Immediate severe post partum Hypertension due to two causes:-

MECHANISMS:-

1) Underlying chronic hypertension.

2) Mobilisation of edema fluid with redistribution into the intravenous compartment

Labetalol and diuretic is effective treatment for both mechanisms.

DIURETICS & HYPER OSMOTIC AGENTS:-

Diuretics can compromise placental perfusion and produces intravascular volume depletion It is used only in case of pulmonary edema. Once delivery occurs there is spontaneous diuresis that usually begins with in 24 hours and disappearance of excessive extra vascular fluid over the next 3 – 4 days.

Hyper osmotic agents infusion shows appreciable intra vascular influx of fluid and escape of intra vascular fluid in the form of edema to vital organs like lungs, brain.

OTHER HYPERTENSION AGENTS:- NHPEP (2000)

Verapamil iv infusion 5 – 10 mg / hour. MAP lowered by 20%

ANATOMICAL CHANGES OF VERTEBRAL COLUMN DURING PREGNANCY

The following are the changes in vertebral column during pregnancy.

1. Uterine enlargement and vena caval compression result in engorgement of epidural veins.
2. Unintentional intravascular cannulation and injection of local anesthetic are more common in pregnant patients.
3. Vertebral foraminal veins which are contiguous with the epidural veins are enlarged and obstruct the pathways for local anesthetic to exit.
4. Enlarged epidural veins may displace cerebrospinal fluid from the thoracolumbar region of subarachnoid space as does the increased intraabdominal pressure of pregnancy. This explains the lowered dose requirement of pregnancy.
5. Subarachnoid dose requirement are also affected by the lower specific gravity of cerebrospinal fluid in pregnant patients as compared with nonpregnant patients.
6. The hormonal changes of pregnancy affect the perivertebral ligamentous structures including ligamentum flavum.
7. Ligamentum flavum may feel lessdense and softer in pregnant woman than in non pregnant woman. It is therefore difficult to feel the movement of needle through the ligamentum flavum.
8. It is more difficult for a pregnant women to achieve flexion of the lumbar spine.progressive accentuation of lumbar lardosis alters the surface anatomy of vertebral column in two ways.

a) A pregnant woman pelvis rotates along the long axis of the spinal column thus the line joining the iliac crest assumes a more cephalad relationship to the vertebral column.

b) Imaginary line might cross the vertebral column at L3-L4 interspace rather than L4-L5 interspace.

9. Less space exists between adjacent lumbar spinous process during pregnancy. Mid line approach is difficult.

10. MRI has shown that apex of the lumbar lordosis is shifted cauded during pregnancy and typical thoracic kyphosis in pregnant women.

ADVANTAGES OF SPINAL ANESTHESIA

Spinal anesthesia has the following

1. Simplicity
2. Rapid onset in cases of fetal distress
3. Dense neural blockade
4. Negligible maternal risk of systemic local anesthetic toxicity
5. Minimal transfer of drug fetus
6. Well suited for emergency cesarean section.

DISADVANTAGES OF SPINAL ANESTHESIA

Though spinal has many advantages over epidural and general anesthesia it has some disadvantages also which are depicted below.

1. Rapid onset of sympathetic blockade produces abrupt severe hypotension
2. Hypotension may not be tolerated by a fetus with evidence of chronic compromised uteroplacental perfusion
   (IUGR, oligohydramnios)
3. When surgery is longer than anticipated supplemental analgesia or change over to general anesthesia may be required.

ADVANTAGES OF EPIDURAL ANESTHESIA

The following are the advantages of epidural anesthesia.

1. Local anesthetic agent can be administered in incremental doses and that the total dose can be titrated to desired level.
2. Slower onset of anesthesia, therefore allows maternal cardiovascular system to compensate for the occurrence of sympathetic blockade, decreased risk of hypotension, decreased risk of reduced uteroplacental perfusion.
3. Can maintain anesthesia regardless of the duration of surgery.
4. Lower extremity muscle pump remain intact and reduced incidence of thromboembolic disease.
5. Opioid or a local anesthetic may be given through a epidural catheter.

DISADVANTAGES OF EPIDURAL ANESTHESIA

Disadvantages of epidural ansthesia is given below.

- Slow onset in case of fetal distress is disadvantageous in case of fetal distress.
- Anesthesiologist must wait for 3-5 minutes before giving therapeutic dose.
- 10-20 minute period is required for adequate surgical anesthesia.
- Post dural puncture headache is more after accidental dural puncture.
- Epidural catheter may migrate into a blood vessel during labour or surgery.
- Increased systemic absorption of local anesthetic results in greater placental transfer o drug to fetus than spinal anesthesia.
- Large amount of local anesthetic required , therefore systemic toxicity may be high.
- Unintentional subarachnoid injection of a large dose of local anesthetic results in high or total spinal anesthesia.

GENERAL ANESTHESIA –DISADVANTAGES

Apart from the common complications like aspiration in general anesthesia for cesarean section there are some unique complications in preeclamptic patients

1. Gross airway edema causing increased incidence of difficult intubation.
2. Hypertensive response to laryngoscopy .
3. Stress response increases intracranial pressure and chances of intracerebral hemorrhage is more.
4. Drug interaction to antihypertensives and magnesium sulphate can occur.
5. Hodgkins et al reported that Systolic blood pressure and PCWP increases significantly in preeclamptic women during intubation , suction , and extubation under general anesthesia
6. Impairment of inter villous blood flow. Thetefore uterine bloodflow is very much reduced

SUPINE HYPOTENSION SYNDROME

IN 15% of patients supine position results in signs of shock including hypotension, pallor, vomiting and decerebration. This is called as supine hypotension syndrome. Inferior venacava compression results in pooling of venous blood and increased uterine and lower extremity venous pressures.

UTERINE PERFUSION PRESSURE = UTERINE ARTERY PRESURE-UTERINE VENOUS PRESSURE

- Uterine artery pressure is decreased secondary to decreased cardiac output.
- Uterine venous pressure is increased with aortocaval compression.
- Supine position should be avoided by pregnant woman at term.

USE OF VASOPRESSORS IN SPINAL ANESTHESIA

EPHEDRINE USAGE IN SPINAL ANESTHESIA

EPHEDRINE:- It is an indirect acting catecholamine that stimulates alpha and beta adrenergic receptors.

MECHANISM OF ACTION

It acts through

1. Endogenous release of norepinephrine- (indirect action)

2. Direct action adrenergic receptors.

USES:

Ephedrine 10 to 25 mg i.v is the most commonly selected sympathomimetic when drug therapy is required to increase blood pressure in the presence of sympathetic nervous blockade produced by regional anesthesia or hypotension due to inhaled or injected anesthetics.

ADVANTAGE OF EPHEDRINE IN PREGNANCY:

Uterine blood flow is not altered when used in pregnant patients under subarachnoid blockade for cesarean section. [24,25,26,27 28, 29]

CARDIOVASCULAR EFFECTS:

1. Increase in systolic and diastolic blood pressure
2. Increase in heart rate and cardiac output
3. Renal and splanchnic blood flow are decreased due to alpha effects
4. Coronary and skeletal muscle blood flow increased.
5. Increased myocardial contractility due to activation of beta 1 receptors.

DISADVANTAGES:

TACHYPHYLAXIS

Second dose of ephedrine produces less intense systemic blood pressure responses than the first dose. This phenomenon is known as Tachyphylaxis.

ETIOLOGY:-

It is due to

1.Persistent blockade of Adrenergic receptors

2.Depletion of Noradrenergic stores

It usage is contraindicated in cardiac patients

REVIEW OF LITERATURE

Wallace et al - 1995 -a prospective study comparing general anesthesia, epidural anesthesia, combined spinal epidural anesthesia for cesarean section in severely preeclamptics showed that hypotension was slightly more in combined spinal epidural group but hypotension was short lasting and revertable.

Hood and Curry et al 1999 –a retrospective study comparing spinal anesthesia and epidural for cesarean section severely preeclamptics showed a 13% decrease in mean lowest mean arterial pressure from baseline in both spinal and epidural groups.

Sushee Visalyaputra et al 2005 - studied spinal versus epidural anesthesia for cesarean delivery in severe preeclampsia and showed that spinal anesthesia produces slightly more hypotension than does epidural anesthesia during the induction to delivery period.The duration of hypotension however was short and there was no difference in neonatal status.

Ramanathan J, et al studied combined spinal and epidural anesthesia with low doses of intrathecal bupivacaine in women with severe preeclampsia. He concluded that CSE with low dose of bupivacaine produces adequate anesthesia for cesarean delivery in patients with severe preeclampsia.

Aya et al. studied spinal anesthesia induced hypotension : a risk comparison between patients with severe preeclampsia and healthy women undergoing preterm cesarean delivery and found that Patients with severe preeclampsia experience less hypotension during spinal anesthesiafor elective cesarean delivery than healthy parturients:

Lee A,Ngan Kee WD made a dose response meta analysis of prophylactic intravenous ephedrine for the prevention of hypotension during

spinal anesthesia for elective cesarean delivery.The results showed that ephedrine can be used safely in preeclamptic women without adverse effects on both mother and fetus because ephedrine increase uterine and placental circulation and therefore the preferred vasopressor in preeclampsia

MATERIALS AND METHODS

Study design : This study was a randomized prospective comparative study.

Study setting and population:

After obtaining Institutional ethical committee clearance, the study was carried out on 84 patients in the operation theatre,Department of Anesthesiology, Tirunelveli medical college, Tirunelveli .

In this study we allocated the patients into two groups

GROUP ---1 is normal group or N group. It constitutes 43 ASA grade I patients coming for cesarean section.

GROUP---2 is preeclamptic group or PE group. It constitutes 41 ASA grade II, ASA grade III preeclamptic patients coming for cesarean section.

SELECTION CRITERIA OF PREECLAMPTIC PATIENTS

Patients with

1. Systolic blood pressure>140mmHg
2. Diastolic blood pressure >90 mmHg
3. Proteinuria
4. Generalized edema were included in the study.

EXCLUSION CRITERIA

The following patients were excluded from the study

1 Gestational diabetes mellitus

2 Inadequate analgesia where supplementation required

3 Blood transfusion required

4 Any intra op complications

5 HELLP syndrome

6 Fetal distress

7 Spinal deformity

8 Height less than 140cms

9 Patients on anticoagulation treatment

10 Antepartum hemorrhage

11 Heart disease

PREOPERATIVE EVALUATION

1 History

2 Clinical examination

3 Urine for albumin &sugar

4 Blood biochemistry including

1. Blood sugar
2. Blood urea
3. Complete hemogram

Just before the operative procedure the following parameters were noted.

1. Height
2. Weight
3. Gestational age
4. Obstetric score
5. Indication for cesarean section

PROCEDURE

Preeclampsia was diagnosed as per Standard diagnostic criteria such as Clinical features , Blood pressure and Laboratory Investigation .

Premedication :- Patients in both the groups received Inj. Metaclopromide 10mg, Inj. Ranitidine 50mg before the procedure.

Both the groups received 10 ml/kg of 0.9% normal saline intravenously before giving Subarachnoid blockade. Subarachnoid blockade was performed using 23 guage Quinckes needle with the patient in right lateral position at L3-L4 inter space and 9mg or 1.8 ml of Hyperbaric Bupivacaine for Lower segment Cesarean Section. Immediately after giving subarachnoid blockade patients were turned supine. A wedge with 10cms height was placed under the right hip to correct uterine displacement. All patients were supplemented with oxygen at 4litres/minute. Level of blockade was T5-T6 in all tne cases . Immediately after delivery of the baby patients in both the groups received Injection . Oxytocin 10 units intravenously and 1mg of Inj.Midazolam intravenously.

Pulse rate, Blood pressure was monitored in both the groups before subarachnoid blockade, immediately after subarachnoid blockade and for every 2 minutes interval for first ten minutes then at every 5 minute interval for a period of two hours.

Temperature, ECG, SPO2, Urine output was also closely monitored in both the groups.

Neonatal APGAR SCORE was recorded at two minute and five minute interval in both the groups.

CRITERIA FOR EPHEDRINE

Inj. Ephedrine was used when patient developed intra operative hypotension with Systolic Bp < 100 mm Hg or fall of 25% of baseline or mean arterial pressure fall of 25% from baseline value.

Injection Ephedrine 6mg was used in incremental doses**.**

RECORDED PARAMETERS FOR STATISTICAL ANALYSIS

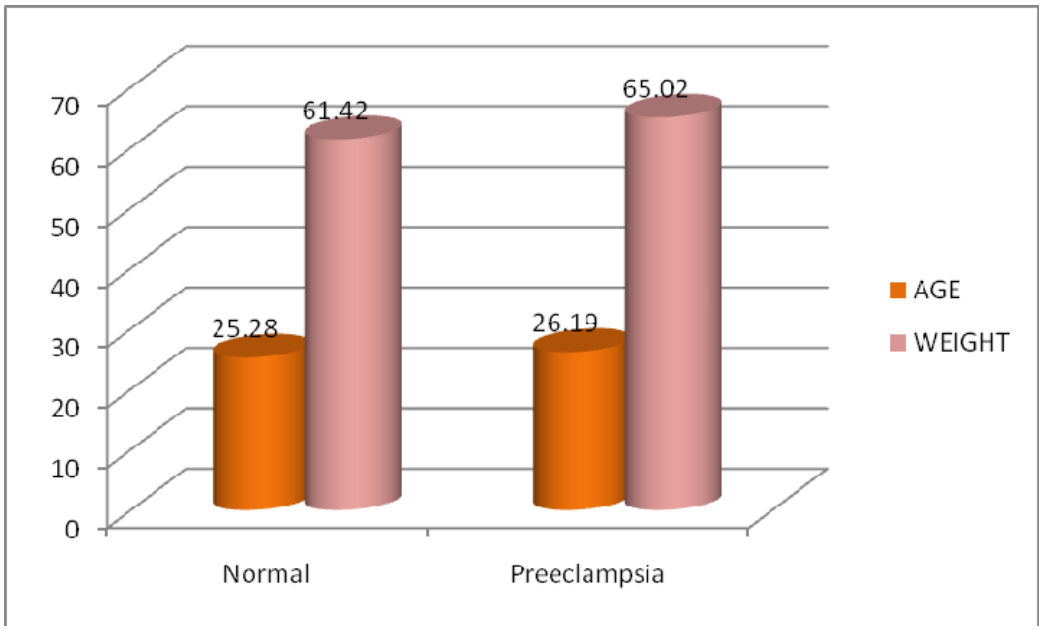
The following parameters were noted both intraoperatively and postoperatively

1. Baseline BP and HR before spinal anaesthesia
2. Maximal fall Percentage of systolic blood pressure and mean arterial pressure
3. Incidence of Bradycardia and Atropine usage.
4. Total dose of ephedrine used
5. Total amount of fluid infused
6. Neonatal Apgar scores@1 min, 5 min
7. Post operative BP two hours after surgery

Student "t" test was used for statistical analysis.

DEMOGRAPHIC VARIABLES

Variable	Normal n=43	Pre.ec N=41	P value	Significance
Age	25.28 ±3.68	26.19 ±3.23	0.230	P>0.05 NS
Weight Kg	61.42 ± 6.53	65.02 ± 6.42	0.014	P<0.05 S



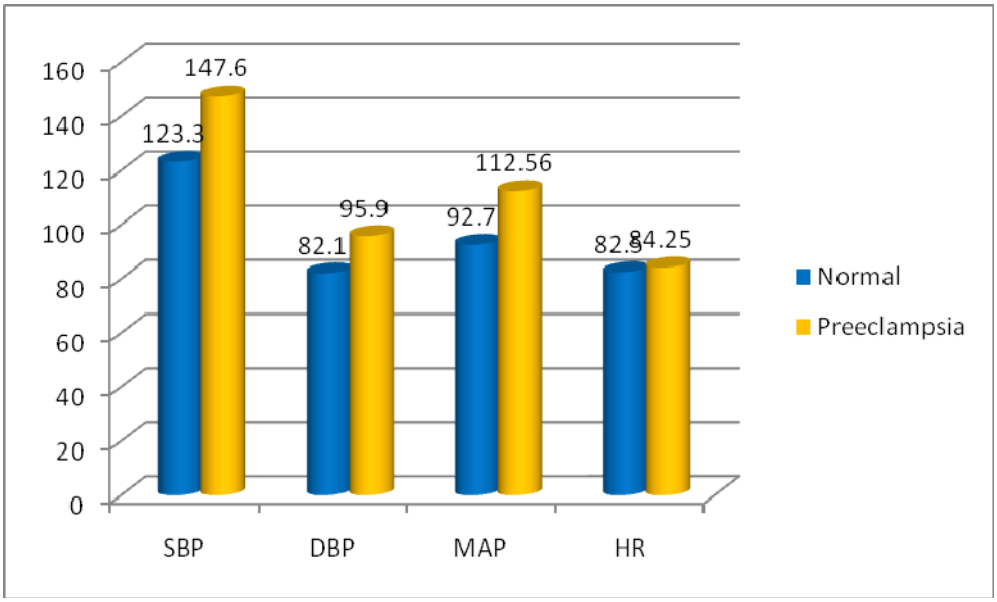
Age in both normal and preeclamptic group is comparable as noted by P value which is not significant Weight was slightly higher in preeclamptic group and P value is less significant.

TABLE SHOWING BASELINE BLOOD PRESSURE AND HEART RATE IN PREECLAMPTIC GROUP AND NORMAL GROUP

	N group N 43	PEgroup N 41	P value	Significance
SBP	123.3 ±12.7	147.6 ± 19.3	0.000	*P< 0.001* HS
DBP	82.1 ± 11.6	95.9 ±13.2	0.000	P< 0.001 HS
MAP	92.7 ±9.7	112.56 ± 15.3	0.000	*P< 0.001* HS
HR	82.5 ±12.25	84.25 ±14.13	0.194	P>0.05 NS

The Systolic blood pressure, Diastolic blood pressure, Mean arterial pressure was higher in preeclamptic group and all the parameters showed a " p " value of < 0.001 which was highly significant. This implies that both the groups were of different entity.

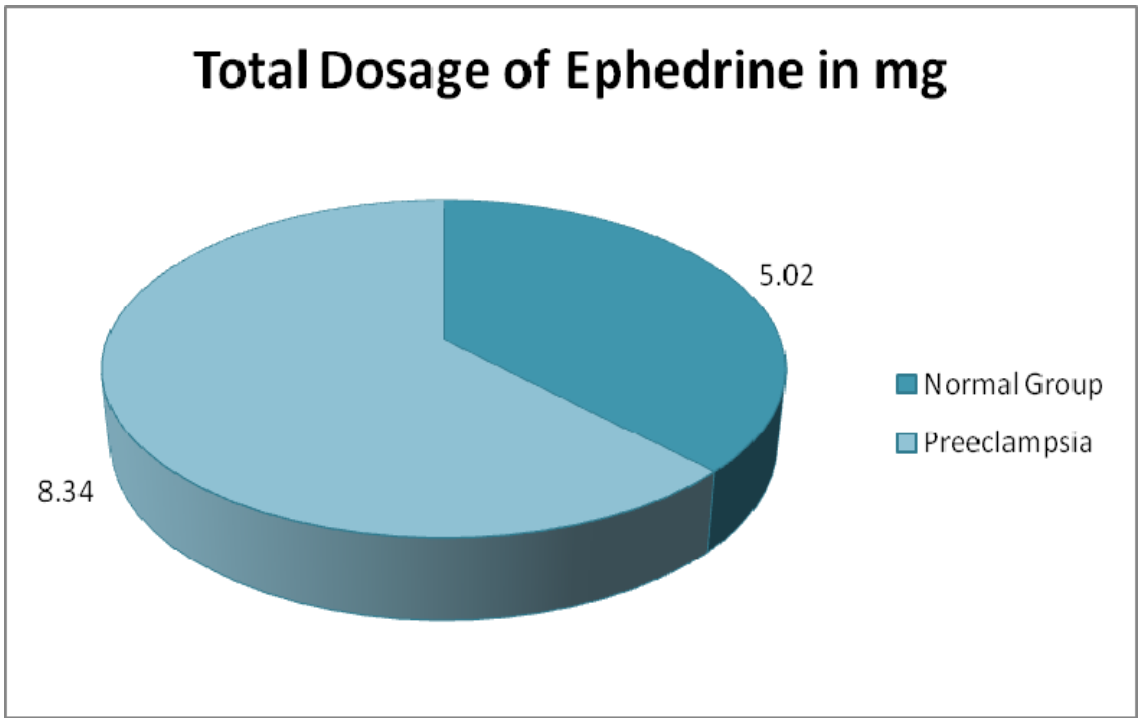
BASELINE BLOOD PRESSURE AND HEART RATE

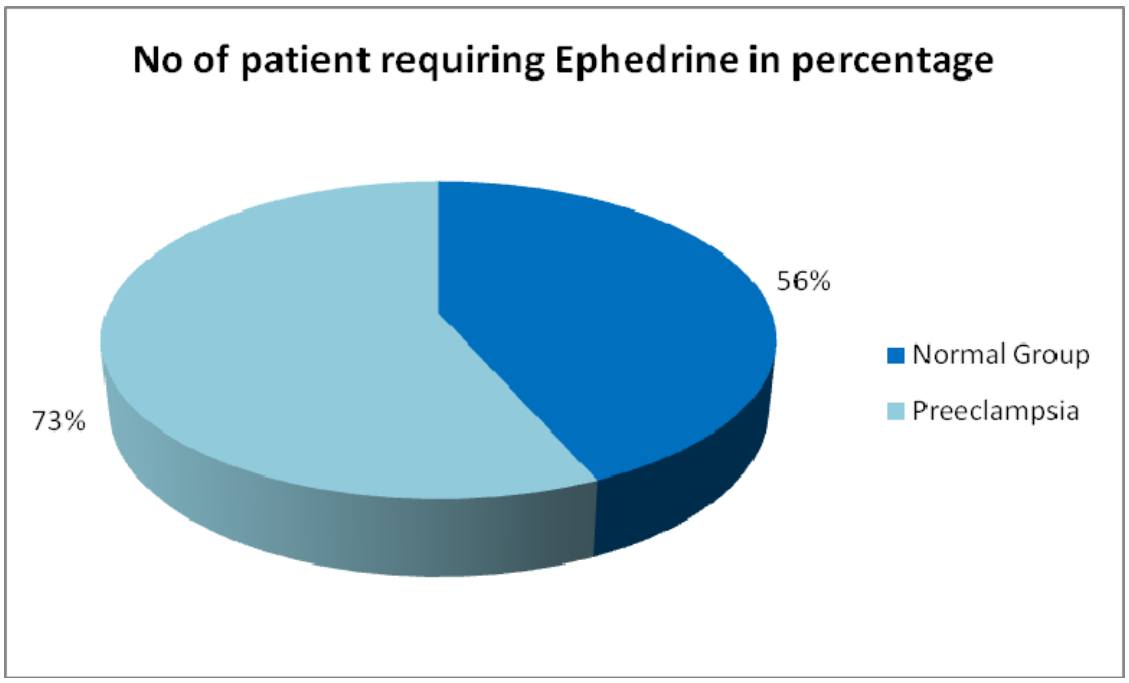


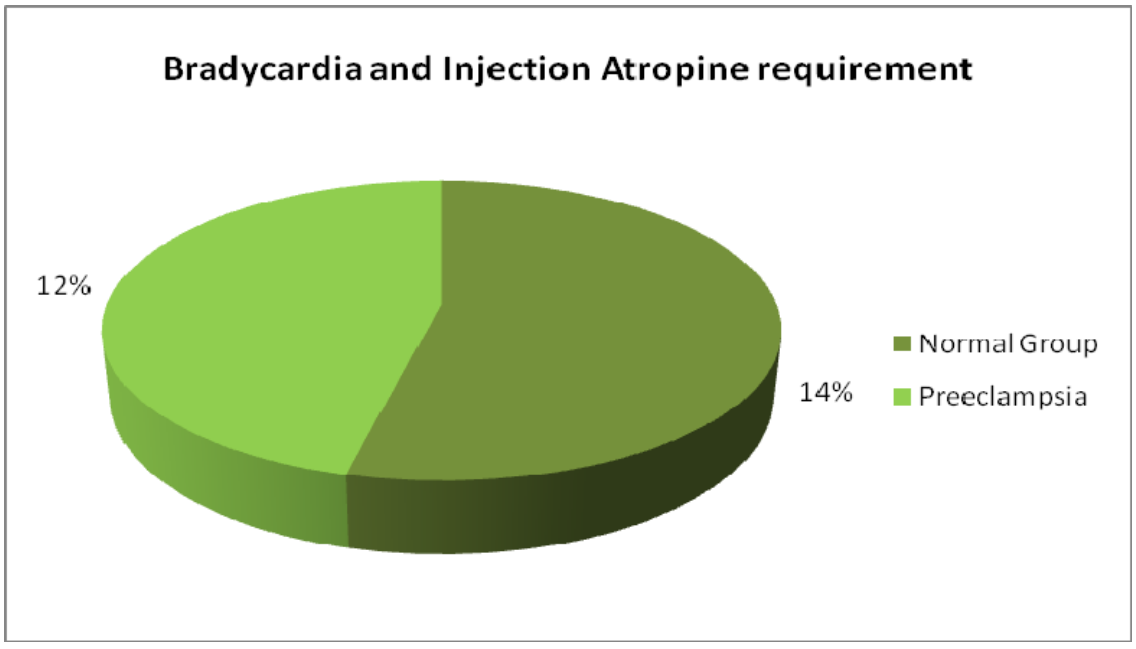
Bar diagram depicting systolic BP, diastolic BP,Mean arterial pressure, Heart rate in Normal and Preeclamptic group

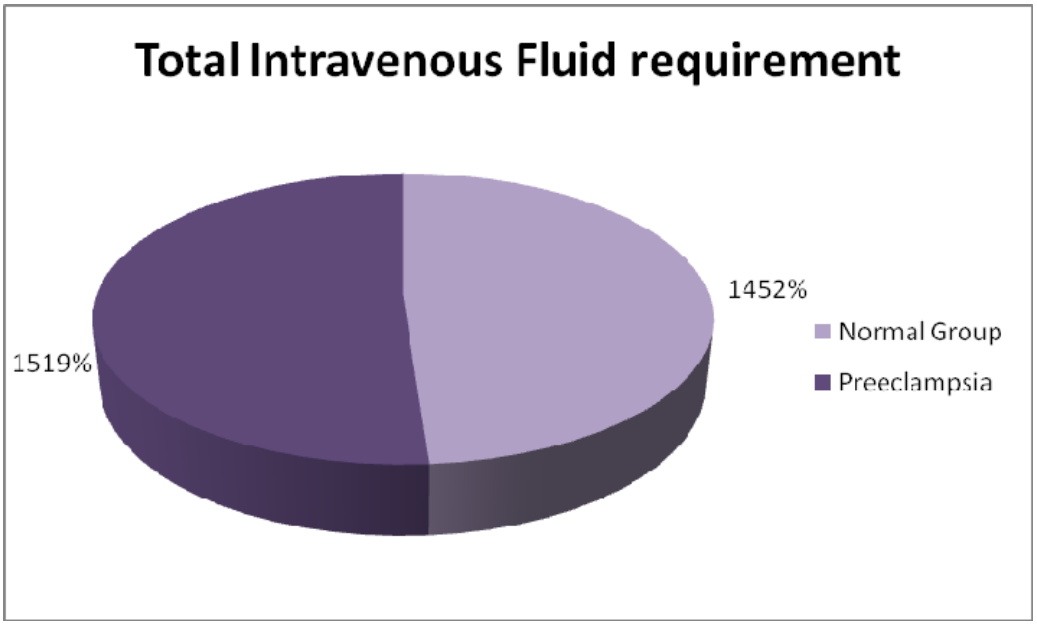
COMPARISON OF EPHEDRINE, ATROPINE, FLUID REQUIREMENT IN BOTH GROUPS

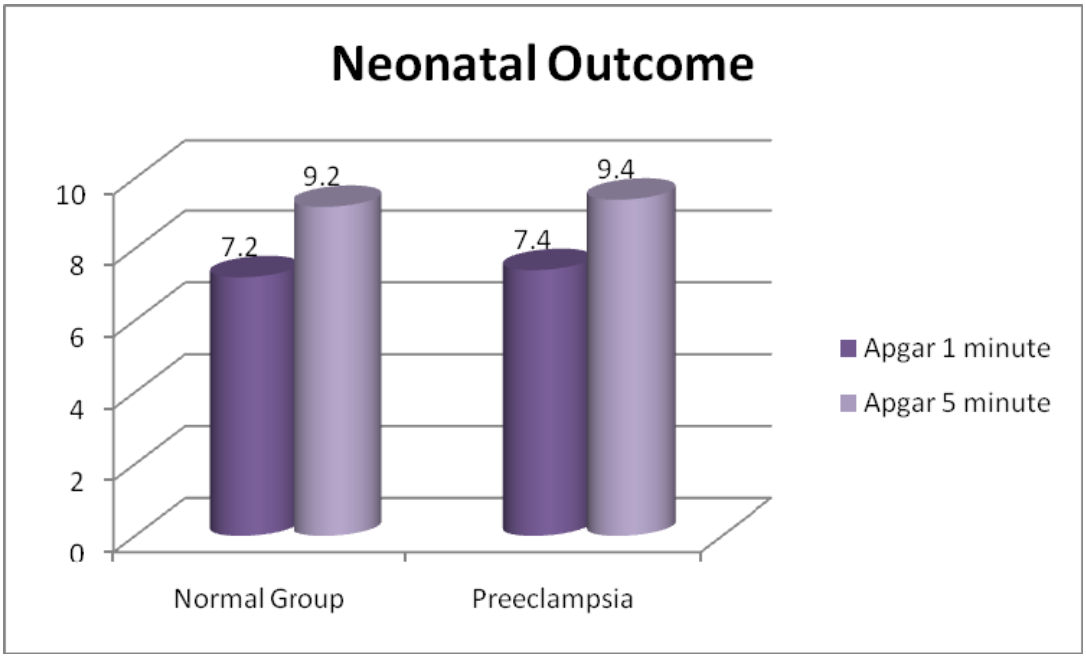
Variables	Normal group	Preeclamptic group	p-value	Significance
Total Ephedrine in mg	5.02 ± 5.39	8.34 ± 6.42	0.012	P<0.05 S
No patient req Ephedrine	24/43 56%	30/41 73%		P>0.05 NS
Bradycardia Atropine req	6/43 14%	5/41 12%		P>0.05 NS
Total IVF	1451.9 ± 118.7	1518.8 ± 151.4	0.026	P<0.05 S
Apgar 1 min	7.2 ± 0.81	7.4 ± 1.03	0.174	P>0.05 N S
Apgar 5 min	9.2 ±0.67	9.4 ±0.7	0.311	P>0.05 NS




No of patient requiring Ephedrine in percentage
56%
73%
Normal Group
Preeclampsia


Bradycardia and Injection Atropine requirement
12%
14%
Normal Group
Preeclampsia



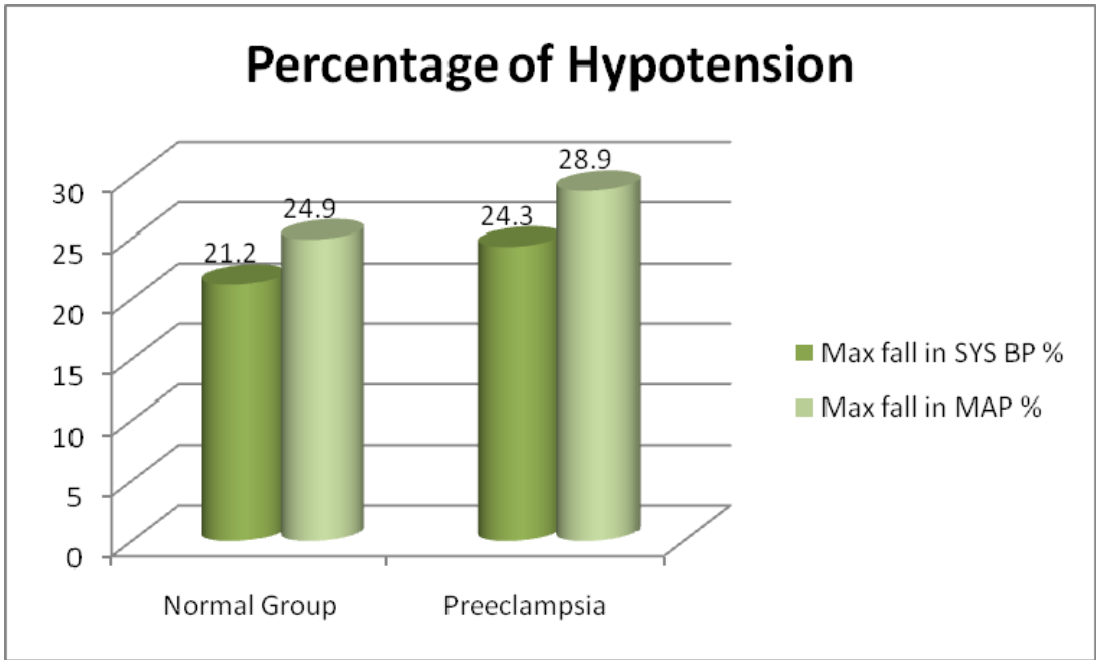


The Total dose of Ephedrine required and Total dose of intravenous fluid required was higher in preeclamptic group . This small percentage of increase is acceptable . The occurance of Bradycardia and Atropine requirement was higher in normal group.

PERCENTAGE OF HYPOTENSION

The maximal fall in systolic blood pressure and mean arterial pressure were noted in both the groups.

	N Group N 43	PEGroup N 41	P value	Significance level
Max fall in SYS BP %	21.2 ±10.7	24.3 ± 10.4	0.186	P>0.05 NS
Max fall in MAP %	24.9 ± 13.4	28.9 ± 11.8	0.148	P>0.05 NS

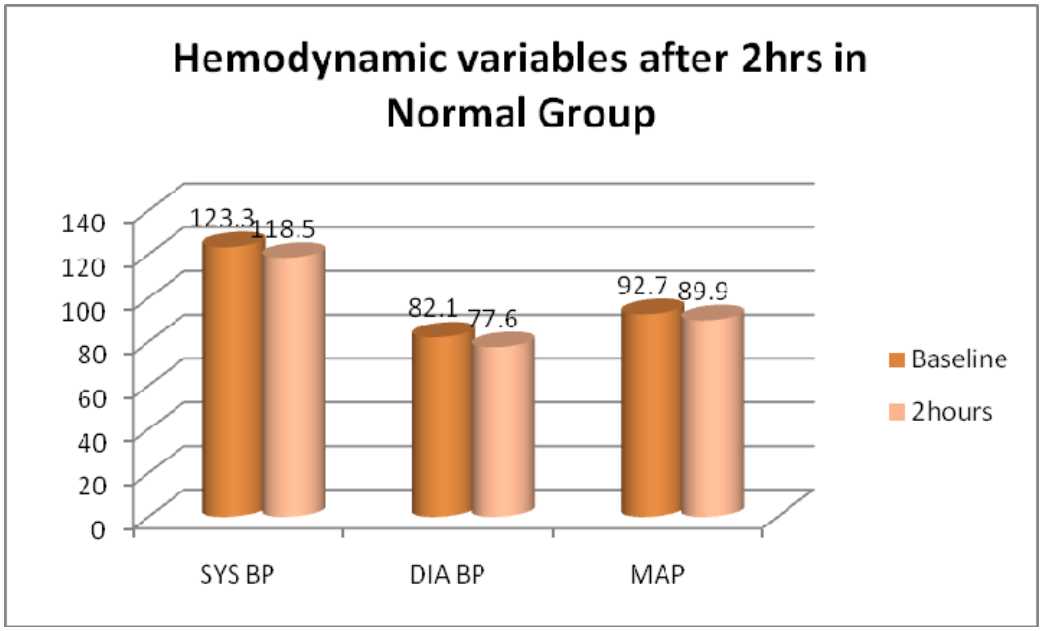


The maximal fall in systolic and mean arterial pressure were noted in both the groups. Fall in systolic blood pressure and mean arterial pressure was the same in both the groups as per the statistical value.

HEMODYNAMIC VARIABLES AFTER TWO HOURS

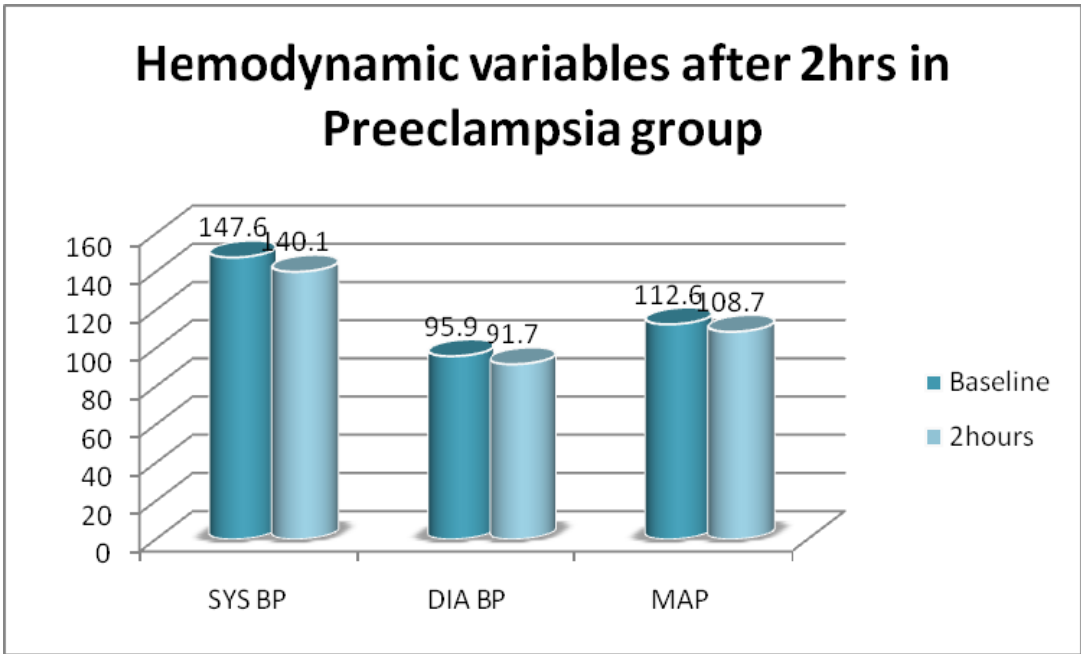
Normal group

	N Group n 43 Baseline	N Group n43 2 h	P value	significance
SYS BP120	123.3 ±12.7	118.5 ±11.1	0.143	P>0.05 NS
DIA BP 120	82.1 ± 11.6	77.6 ±10.8	0.154	P>0.05 NS
MAP	92.7 ±9.7	89.9 ±10.6	0.151	P>0.05 NS



PREECLAMPTIC GROUP

Variables	PE group n 41 Baseline	PE group N 41 2 h	P value	Sinificance
Systolic BP	147.6 ± 19.3	140.1 ±15.3		P>0.05 NS
Diastolic BP	95.9 ±13.2	91.7 ±12.5		P>0.05 NS
Mean arterial pressure	112.56 ± 15.3	108.7 ±16.2		P>0.05 NS



The Systolic blood pressure, Diastolic blood pressure and Mean arterial pressure returned to the baseline values in both the groups.

RESULTS OF THE STUDY

In this study, in normal group the mean systolic BP is 123 mmHg , mean diastolic BP was 82mmHg , mean arterial pressure is 92 mmHg. In pre eclamptic group systolic BP, diastolic BP, mean arterial pressure is 147, 95, 112 mmHG respectively. Its P value is less than 0.001 which is highly significant. This indicates that both the groups are of different entity.

The maximum fall in systolic BP, MAP is 21.2% and 24.9% in normal group compared to 24.3 % and 28.9 % in preeclamptic group . P value is also not significant indicating that there is no significant hypotension.

Regarding vasopressor requirement the amount of Ephedrine used intra operatively in preeclamptic group is 8.34 mg compared to 5.02 mg in normal group. P value is less than 0.05 which is significant .This 3mg of increased Ephedrine requirement is used without any adverse effects to the patients. Percentage of patients requiring Ephedrine is 73% in preeclamptic group compared to 56% in normal group.

Total Intravenous fluid infused is 1518 ml in preeclamptic group compared to 1451 ml in normal group. Its P value is not significant.

Brady cardia occurrence in normal group is 14% compared to 12% in preeclamptic group. Hemodynamic variables after two hours returned to baseline values in both the groups .

APGAR score at 1 minute and 5 minute interval is 7.2 and 9.2 in normal group compared to 7.4 and 9.4 in preeclamptic group and reveals no adverse neonatal out comes.

DISCUSSION

In this study patient selection criteria were determined in an attempt to select only severely hypertensive patients whose obstetric therapy least complicated interpretation of blood pressure response to spinal anesthesia. Labouring were excluded because they may be less likely to suffer hypotension during regional anesthesia for cesarean section. Also patients with diabetes, heart disease, patients requiring intraoperative blood transfusion were excluded since their cardiovascular status may be influenced by their disease pathology.

Dosage of Hyperbaric Bupivacaine used in this study is 9mg .Dosage used in Wallace et al, Aya et al ,Hood and curry et al were 11.4 mg,13.5 mg,11mg respectively. This decreased dosage used in this study may account for decreased hypotension in this study. Factors such as management of Intravenousfluids , Antihypertensive therapy, Ephedrine usage may contribute to the differences observed in the decrease in blood pressure.

Ephedrine usage was 8.75 to 10mg in Aya et al study and it was 8.34 mg in this study. Number of patients requiring ephedrine is 23%, 32% ,72%, in Hood and curry et al , Wallace et al , Sushee visalyaputra et al. In this study it was 73% which is comparable to other studies

Intra venous fluid required intraoperatively for spinal anesthesia was 1780 ml in Ayaet al and 2255 ml in Wallace eal study. In this study we have used 1518 ml which is comparable to the above studies

This study shows that spinal anesthesia for cesarean section in severely preeclamptic patients causes slightly more reduction in blood pressure. The duration of hypotension was however was short and there was no difference in neonatal status.

In the prospective study by Wallace et al comparing general anesthesia, epidural and combined spinal epidural anesthesia for cesarean delivery in severely preeclamptic patients ,the mean lowest systolic arterial pressure and diastolic arterial pressure values after CSE technique were similar in this study.

A retrospective study by Hood & Curry et al compared spinal anesthesia and epidural anesthesia for severely preeclamptic patients for cesarean delivery. A similar study comparing spinal and epidural anesthesia was done by sushee vishalyaputra et al . The results of these studies is given below.

Study	Percentage of fall in MAP in spinal anesthesia	Percentage of fall in MAP in epidural anesthesia
Sushee visalyaputra et al	51%	23%
Wallace et al	25%	25%
Hood and curry et al	13%	13%
This study	24%	-

In this study the fall in Mean Arterial Pressure is comparable to other studies. There is no significant hemodynamic changes induced by spinal anesthesia in preeclamptic patients and neonatal out comes assessed by APGAR score was normal as compared other studies.

SUMMARY

This study summarises that spinal anaesthesia in Normal and Preeclamptic groups

1) Does not alter the hemodynamic variables. Although the incidence was slightly more frequent in preeclamptic group , the hypotension is short lived and easily correctable without imposing any danger to both the mother and the fetus.
2) Neonatal outcome was same in both the groups
3) Vasopressor requirement and Total intravenous fluid infused was marginally raised but adding 3 mg of Inj.Ephedrine in addition is quiet acceptable.

CONCLUSION

1) Spinal Anesthesia does not produce gross haemodynamic changes in Pre eclamptics
2) SAB can be safely practiced in pre eclamptic for LSCS
3) The benefit of Rapid, dense and reliable SAB over epidural anaesthesia should be considered for preeclamptics undergoing cesarean section.

REFERENCES

1. Ramanthan J,Coleman P,Sibai B, Anaesthetic modification of haemodynamic and neuroendocrine stress response to cesarean delivery in women with severe preeclampsia Anesth Analg 1991;73:772-9

2. Ramanathan J, Vaddadi AK, Arheart KL. Combined spinal and epidural anesthesia with low doses of intrathecal bupivacaine in women with severe preeclampsia: a preliminary report. Reg Anesth Pain Med 2001;26:46 –51.

3. Hood DD, Curry R. Spinal versus epidural anesthesia for cesareansection in severely preeclamptic patients: a retrospectivereview. Anesthesiology 1999;90:1276–82.

4. Aya AGM, Mangin R, Vialles N, et al. Patients with severepreeclampsia experience less hypotension during spinal anesthesiafor elective cesarean delivery than healthy parturients: aprospective cohort comparison. Anesth Analg 2003;97:867–72

5. Santos AC, Birnbach DJ. Spinal anesthesia in the parturient with severe preeclampsia: time for reconsideration. Anesth Analg2003;97:621–3

6. Visalyaputra S, Rodanant O, Somboonviboon W, et al. Spinalversus epidural anesthesia for cesarean section in severepreeclampsia: a prospective randomized, multicenter study.Anesth Analg 2005;101:862– 8.

7. Newsome LR,bramwell RS, curling PE.severe preeclampsia hemodynamic effects of lumbar epidural anesthesia.anesth anaiog 1986.

8. Hypertensive disorders in pregnancy .in Cunningham FG Gant NF ,Leveno KJ,eds.Williams obstetrics ,21st edition .newyork:MCgraw hill,2001

9. Riley ET,Cohen SE,Macario A,et al.spinal versus epidural anesthesia for cesarean section: acomparison of time efficiency,cost, charges and complications.anesth analog 1995.

10. Malinow AM. Spinal anesthesia in preeclamptic patients. “supportive”

evidence. Anesthesiology 2009.

11. Karinen J,Rasanen J,alahuhta S et al .maternal and utero placental hemodynamic state in preeclamptic patients during spinal anesthesia for caesarean section.Br.J anesth 1996.

12. Assali NS, Prystowsky H. Studies on autonomic blockade. I. Comparison between the effect of tetramethylammonium chloride (TEAC) and high selective spinal anesthesia on blood pressure of normal and toxemic pregnancy. J Clin Invest 1950; 29: 1354–66.

13. Lechner RB, Chadwick HS. Anesthetic care of the patient with preeclampsia. Anesth Clin North Am 1990; 8: 95–114.

14. Cheek TG, Samuels P. Pregnancy-induced hypertension. In: Data S, ed. Anesthetic and obstetric management of high-risk pregnancy. St Louis: Mosby, 1996: 386–411.

15. Maresh M, James D, Neales K. Critical care of the obstetric patient. In: James DK, Steer PJ, Weiner CP, Gonik B, ed. High risk pregnancy: management options. London: WB Saunders, 1999: 1291–1321.

16. Wallace DH, Leveno KJ, Cunningham FG, et al. Randomized comparison of general and regional anesthesia for cesarean delivery in pregnancies complicated by severe preeclampsia. Obstet Gynecol 1995;86:193–9.

17. Dyer RA, Farbas J, Torr GJ, et al. Prospective, randomized trial comparing general with spinal anesthesia for cesarean delivery in preeclamptic patients with a nonreassuring fetal heart trace. Anesthesiology 2003;99:561–9.

18. Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. Am J Obstet Gynecol 2000;183:S1–22.

19. Hays PM, Cruikshank DP, Dunn LM. Plasma volume determination in normal and preeclamptic pregnancies. Am J Obstet Gynecol 1985;151:958–66.

20. Sharwood-Smith G, Clark V, Watson E. Regional anaesthesia for caesarean section in severe preeclampsia: spinal anaesthesia is the preferred choice. Int J Obstet Anesth 1999;8:85–9.

21. August P, Lindheimer MD. Pathophysiology of preeclampsia. Hypertension 1995;142:2407–26.

22. Khalil RA, Granger JP. Vascular mechanisms of increased arterial pressure in preeclampsia: lessons from animal models. Am J Physiol Reg Integrative Comp Physiol 2002;283:R29–45.

23. Visser W, Wallenburg HC. Central hemodynamic observations in untreated preeclamptic patients. Hypertension 1991;17: 1072–7.

24. McKinlay J, Lyons G. Obstetric neuraxial anaesthesia: which pressor agents should we be using? Int J Obstet Anesth 2002; 11:117–21.

25. Harrop-Griffiths W, Thomas DG. Ephedrine is the vasopressor of choice for obstetric regional anaesthesia. Int J Obstet Anesth 2002;11:275– 81.

26. Lee A, Ngan Kee WD, Gin T. A quantitative, systematic review of randomized controlled trials of ephedrine versus phenylephrine for the management of hypotension during spinal anesthesia for cesarean delivery. Anesth Analg 2002;94:920–6.

27. Ngan Kee WD. Obstetric neuraxial anaesthesia: which vasopressor should we be using (letter). Int J Obstet Anesth 2003;12: 55–64.

28. Ngan Kee WD, Khaw KS, Ng FF. Comparison of phenylephrine infusion regimens for maintaining maternal blood pressure during spinal anaesthesia for caesarean section. Br J Anaesth 2004; 92:469 –74.

29. Casey BM, McIntire DD, Leveno KJ. The continuing value of the Apgar score for the assessment of newborn infants. N Engl J Med 2001;344:467–71.

30. Redman CW,sergeant IL.Preeclampsia,The placenta and the maternal systemic inflammatory response :a review.placenta 2003.

31. Sibai BM :pulmonary edema in severe preeclampsia analysis of 37 consecutive cases obstet gynec 1987

32. Talledo OChesley LC :rennin angiotensin system in normal and toxemia of pregnancy.obstet analog 1968

33. Gant NF ,Daley GL , Chand S et al –A study of Angiotensin II pressor response throughout primi gravid pregnancy 1973

PROFORMA

STUDY – Spinal anesthesia in Preeclamptic Vs Normal patients for LSCS

Name : **Age:** **Ht:** **Wt:**

I.P.No: **Unit:** **Date:**

Obstetric score: **Gestational week:**

Type of surgery: Elective / Emergency **ASA status:**

Indication:

PIH History: **Duration:**

Swelling of legs
Oliguria
Headache
Visual disturbances

Drug history: **dose:** **duration:**

Nifedepine
Alpha methyl dopa
$MgSO_4$
Aspirin
Others

Investigations:

Hb : **Urine albumin :**
Blood Sugar : **Sugar :**
Urea : **deposits :**
Creatinine :
Uricacid : **BT :** **CT :**
Platelet count : **PT :** **aPTT :**
LFT :

O/E

Anaemia : **Icterus :** **Edema :**
PR : **BP :**
Fundus Examination :

Preparation :

Antiemetic prophylaxis
Inj. Ranitidine 50mg i.v.
Inj. Metoclopromide 10 mg i.v.

I.V.Infusion of 0.9% NS – 10ml / kg

Anaesthesia – SAB

Position : Rt lateral
Space : L3 – L4
Needle : 23G
Drug : 0.5% Bupivacaine Hcl 1.8ml

Intra op:

Wedge 10cm ht under right hip
Oxytocin used
Sedation : Inj. Midazolam 0.05mg/kg i.v.
Shivering / vomiting

MAP < 30% of initial value treat with ephedrine 6 mg incremental doses

Parameters monitored

TIME	PR	SBP	DBP	MAP	SpO2	Ephedrine used
Prior to SAB						
Imm. after SAB						
2 min						
4 min						
6 min						
8 min						
10 min						
15 min						
20 min						
25 min						
30 min						
35 min						
40 min						
45 min						
50 min						
60 min						
90 min						
120 min						

Total ephedrine

Neonatal outcome
APGAR score: 0min 1min 5min

Total fluids infused:
Urine Output:

Signature